Thesis on
Alcoholic Neuritis,
its Clinical Features and Pathology.

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Prefatory Notice:

For this thesis I have been unable to collect material during a period of study in Prag. My clinical observations I have made in the wards of the Royal Infirmary allotted to Professors von Jaksch and Prinzen, and in the Pathological Institute attached to the University of Prag. Through the kindness of Professor Czerny in each case I have been enabled to carry out a more or less complete pathological examination. The clinical history of the case of the child I have received from Professor Ganghofner, of the Children's Hospital, Prag.

Though I disapprove of the term Multiple Alcoholic Neuritis and consider the Science to be a more or less pure parenchymatous degeneration, I feel compelled to write under that heading, since most of the writers on the subject have described the affection under the same title and it has become too firmly established to allow of alteration.
Clinical Features of Alcoholic Neuritis.

An American, James Jackson, seems to have been the first, to have made any mention of alcoholic neuritis clinically, and in our country, Wilks was probably the first to give anything like the clear account of the clinical features of the disease, but, with the exception of Dreschfield, Bezzard, Broadbent, Ross, Hadden, Frimley, Thorne and Payne, few English writers have attempted to describe the disease since Wilks' first publication.


On the Continent, on the other hand, as the literature on the subject which is already massive shows, it appears to have excited considerate more attention. Leyten, Mosch, Lancereaux, Schulz, Pichet et Vaillant, Oppenheimer, Dejerine, Eichhorst, Sièmerling, Strümpell, and Bernhardt are the names which are most prominent.


In this connection it is remarkable to note that Magnus Huss in his classical work on Alcoholismus Chronicus, and also Magnan make no special reference to the disease, and that the former should have stated that the symptoms of Alcoholismus Chronicus are unaccompanied by any obvious structural diseases changes.

Alcoholic Neuritis is unquestionably the commonest of all the forms of Multiple Neuritis, and results from a long continued excess of the stronger and more spirituous intoxicants. Gover's states, that it is probably three times more frequent among women than among men. Wickr is of a similar opinion. In England this would seem to hold good as most of the cases that have been described by English writers have certainly been females, but on the continent however it does not hold, and of all the cases which I have seen none have been females. The fact that women in England are more given to the excess we alcoholic indulgence than those on the continent makes it the more difficult for me to accept the affirmation that the female sex offers.


Translated by Granfield.


4. Wickr (S.) 'Lec. cit.'
a special predisposition to this disease.

In most cases the symptoms supervene gradually and insidiously, but they may come on more suddenly being excited by chills, exposure to cold and similar disturbing causes, the system of the individual having been previously brought into a ripe unstable condition by long-continued excesses.

In a multiple, widespread disease like Alcoholic Neuritis it is but natural that the symptoms should be manifold and intermittent, and yet certain symptoms so in certain cases predominate and so allow of a judicious classification and of greater freedom in description. Brecht first divided cases of Alcoholic Neuritis into:

1. Ataxic,
2. Paralytic and atrophic,

and his observations have since been confirmed by many writers on this subject; a third class has been suggested to include those cases where sensory anomalies predominate, and this group seems to me to have also a justifiable existence.

The first signs of the disease consist in a diminution of the delicate sense of touch in the hands and feet, sensations of pins and needles in the limbs, numbness and a feeling of heatness in the fingers and toes, not unfrequently
accompanying by cold blue extremities, this is termed to a condition of vessel cramp comparable to that which is said to occur in Ragnaud's disease, after a time shooting lancinating pains sometimes running from the feet up to the hip and complaints of, and cramp, particularly in the muscles of the calf and apparently coming on most frequently and most severely during the night, sometimes after a few weeks and sometimes not till months after the first symptoms have manifested themselves. Paroxysms occur in the muscles of the extremities, as a rule first in the lower extremities, this paresis gradually advancing and attacking other muscles may finally lead to complete paralysis. The gait soon becomes unsure and unsteady and all fine movements of the hand etc. are rendered impossible. Further, as the paresis advances, so the muscles affected begin to atrophy, and this atrophy may assume such an extreme degree that virtually little else may remain in the lower leg and forearm beyond skin and bone.

As already mentioned, of the two extremities the leg generally shows the first signs of the disease and suffers most severely, but in both the upper and lower extremities it is the extensor muscles which first become paralysed.

1. Potent symptoms of alcoholic Paralysis.

_Lancet_ 1. 23. p. 1125 - 1883
and which atrophy must, namely in the leg, the muscles in the foot itself, the Atonics, the Extensor Anterior, the Extensor Communis Digi, and to a much lesser extent the Quadriceps Extensor, and in the case of the upper extremity, the Extensor Communis Digi, the Extensor Minimi Digiti, the Extensor Carpi Radialis Longus and the Extensor Carpi Ulnaris. As a least Paralysis of the supinator are less affected than the extensors and the paralysis similarly occasions the peculiar wrist drop (writing position) and foot drop, in alcoholic cases, however, the atrophy and paralysis are more widely distributed and less well defined.

The muscles of the trunk and of the face and neck do not partake to any highly appreciable extent in the degeneration, but the diaphragm does not escape and indeed, in a later stage of the disease a loss of this muscles function often hastens the fatal issue. Though affection of the vagus the heart seems to partake also in the general muscular failure, this is indicated clinically by a high degree of tachycardia and by auscultatory signs of feeble beat action.

Paralysis of certain ocular muscles has been described by several writers and in the late stages of the disease seems to be not particularly uncommon and this in spite of
the fact that no pathological change has been found in the nerves which would satisfactorily account for the paralysis of the muscles which they supply, Thomsen mentions three cases of Abducens Paralysis and two of Ploșeic. Schulz cites a case of a Strabismus Convergens from Double Abducens Paralysis. Suckling has a case of Ploșeic with slight right Externl Strabismus, in the same case the vertical eye movements were abolished but the lateral movements were partly impaired. Leidenfrost also has a case of Abducens Paralysis.

With the exception of the pharyngeal muscles supplied by the glossopharyngeal nerve which in severe cases unmistakably are paralysed and give rise to difficulty in swallowing, the muscles along the alimentary canal do not appear to suffer much. The tongue has, so far as I can discover never been described as paralysed or wasted. Along of the lower intestinal tract.

1. Thomsen (S.), Zur Klinik und pathologischen Anatomie
2. Schulz, loc. cit.
3. Suckling (W.), OphthaHmoplegia Externa Due to
and retention of urine not infrequently occur in the later stages of the disease but it is probable that these are not the result of a genuine paralysis but occur in connection with the generally deplorable condition or, since they are most frequently present in those who at the same time show signs of alcoholic dementia, they may be due to personal neglect rather than to intestinal or retrovors paralysis.

The sensory changes to be observed in alcoholic neuritis are also highly important and various. Tierowst states that the existence of sensory changes, if only to a slight extent, is one of the most constant signs of the disease. Though these anomalies exist as a rule in combination with the motor impairment, they may yet exist alone and may predate, with the exception of the sensations of numbness, tingling etc., which have been already mentioned as occurring in the earlier stages, signs of serious sensory impairment do not present themselves until somewhat late in the disease, namely when the atrophy and paralysis have commenced or are fairly well advanced. Anesthesia is the commonest cutaneous anomaly and most generally occurs in patches on the feet, the legs and the forearms, and especially about the

fingers and wrists, it is rarely or never widespread; in many cases, also, the rule of antithesis of painful hyporeflexia in this region is strengthened; paraesthesiae are frequently complained of, and patches of cutaneous hyperesthesia are sometimes discovered; instances of double sensation have also been described ("Dejerine, "; "E. Remak").

With regard to the reflexes, the various cutaneous ones are often not affected but can in the later stages, though this is not the rule, be found wanting.

The Patellar, the Ancon Reflexe (Patailas, Ancon ouchi and Encke) as a rule are not to be elicited and in most cases disappear early in the course of the disease, in two of my cases, however, the patellar reflex could be elicited though the patient was paralysed to such an extent that locomotion was impracticable.

"Vitkowski has made similar observations.
"Rose has observed exaggerated tendon reflexes in

allied forms of multiple neuritis, more particularly in that from product by Chronic Lead Poisoning. Strümpell and Moebius also cite cases in which exaggerated reflexes were present.

With the muscular paralysis, the muscular sense generally fails. An important diagnostic feature in the disease is the existence of severe pain on pressure excited in the paralyzed muscle, and on the trunks of the nerves supplying the same. Both of these phenomena it is extremely difficult to explain. 2 Senator and 3 Siemens's view that the disease is really a primary myositis may go to explain the muscle tenderness; 4 Jowett advanced the view that it is more likely to be due to change in the nerves of the sheath (the 'nerve' nervous') than to an inflammatory inflammation. These severe muscular pains are also felt when the muscles are voluntary exercised, and it is often difficult to determine, especially in a patient who at the same time is suffering from psychical aberration, whether the mobility...

to move or cease by actual paralysis or only exists through fear of movement.

With regard to the special senses nothing of importance is to be described, anamnesis is occasionally found and a slight degree of blurring of the ephelis papilla. Sharkey, further, in one of his cases in which the ophthalmoscopic examination was conducted by Nettlefish, found a form of retinitis which differed essentially from albuminous retinitis and which Sharkey proposes to call an albuminous retinitis.

As one would naturally be led to expect the electrical changes in this condition are considerable. In the whole the rule holds good that the diminution in the electrical excitability is proportionate to the degree of atrophy and paralysis in the muscles stimulated; (Baur, E. Remak, Oppenheimer, Bernhard) for instance in examining an early paralysed muscle it is common to obtain a partial reaction of regeneration, that is to say, there is a slight reaction to the Paradie current directly and indirectly applied, while with the Galvanic current direct stimulation of the muscle produces consistently more contraction than indirect, this phenomenon is explainable by the fact that a considerable number

of undegenerate fibres still remain for the conduction of the stimulating current, as the paralysis and atrophy in these muscles advances, so the regenerative reaction becomes more complete and in some cases does finally become quite complete, this termination is by no means the rule and agree with the pathological fact that it is rare to find a complete destruction of any given nerve, in the other hand as Kahler and Pick, E. Remak, Bernhardt and Oppenheim have shown, it is possible to get the reaction of regeneration even in nerves in which throughout the whole course of the disease have preserved their function and never given rise to motor or sensory paralysis in the parts supplied by them; this is certainly a most puzzling result. The electrical examination in these cases is both extremely difficult to carry out and apt to be unreliable in account of the very severe pain, which the application of even a slight


Another most remarkable feature in this disease is the extraordinary degree of ataxy which sometimes develops and often leads to great difficulty in distinguishing it from true cerebellar atrophy.

Watson cites the term Pseudoataxia for alcoholic and "Fischer ni de" for the first time gave a description of some cases in which "potatoes" were attacked with symptoms of ataxia and atrophy, and showed at the same time the reaction of degeneration, and failure of the tendon reflexes; since that time the matter has excited a great amount of attention and been repeatedly described (Lanceareus, Moebi, Dickfelig, Krich, Strumpell, Bernhardt, Heft, Schult, Christiansen).

2. Lanceareus (loc. cit.)
   Charité Annalen. 1854.
4. Dickfelig. loc. cit.
   Deutsche Mediz. Zeitung. 1854.
   Neuro. Centrale. i4 p. 4184.
10. Christiansen. loc. cit.
and many others; but in most cases no change has been found in the Spinal Cord resembling that occurring in Cabel Dorsalis to account for the ataxy. In addition to this ataxic fact, the Bracht-Chromberg phenomenon and the loss of the patellar reflexes, it is common to find a marked degree of ataxy and neuromatous in the upper extremities. 

Pétrie describes a remarkable case in which there were many of the signs of Cabel, viz, cramps, shooting pain, griddle pain, ataxy, paraesthesia, Chromberg's phenomenon, impotence, polyuria, incontinence of urine and pasties crises, and yet, at so little were the knees just about, and after the autopsy the spinal cord was seen to be perfectly healthy. 

Gorzo and Fusari mention the occurrence of laryngeal and vesical crises, these they ascribe to a neuritis of the sympathetic nerves. 

Cherubs as to the cause of the ataxic phenomena in Alcoholi neuritis are numerous, some writers holding it to be the result of a pure central change, others looking on it as of entirely peripheral origin; on the other hand, some regard it as due to impairment of motor innervation while others consider it to be of sensory

1. Pétrie. Sur un cas de Pseudotable.
   Arch. de Neurologie 63, N° 45, pp. 76-86.
The following seems to distinguish alcoholic pseudo-attacks from true attacks. The existence of the former of a rapidly increasing intensity, of some amount of fever, and of different electrical reactions (in the former, as I have already stated, there is a pronounced diminution of electrical excitability, with or without quantitative changes, now in other dorsal) according to End, there is increased excitability in the earlier stages, while in the later stages, there is a distinct diminution without, however, any quantitative changes) further, in pseudo-attacks, the Argyll Robertson phenomenon, gastric and laryngeal crises, and the gristle pains are rare, again. Charcot lays stress on the character of the gait, pointing out that in alcoholic neuritis, the gait is of a peculiar stamping nature ("steppage") dependent upon the paralysis of the Peronei muscles. It is interesting in this connection to note, as Leyden has pointed out, that Exquisite ataxia may also develop in other forms of neuritis, viz. in those resulting from Diphtheria, Syphilis.

1. Charcot. La paralysie alcoolique.
   Gaz. d. Hop. 62.

Smallpox and other infectious diseases also from arsenic poisoning (Dana, 2. Seeljmiiller, 3. Faltenhem, Kovac) phosphorous and lead poisoning, and amongst cachetic neuvius in diabetes (Charcot, 6. Pryse).

Vasomotor and trophic changes, in the later stages of the disease, are fairly common, the most common being the formation of acute ulcers in hallucial and vesicular eruptions. Hadden in addition mentions the occurrence of syphilitic skin, ulcerative and insufficient healing of the foot, arrest of the growth of the nails, and falling matter out of the nails, as well as profuse

\[\text{(1) Dana. On pseudotales from arsenical poisoning etc. Braii 1886-88.}\]


\[\text{(5) Charcot. Sur un cas de paraplegie diabetique. Arch. de neurologie. 1890}\]


\[\text{Neurul. Centralhall. 80 p. 866.}\]

\[\text{(7) Hadden. Discussion on Dr. Payre's address in the mortification.}\]

\[\text{Anat. & Path. of alcoholic manic. chron. Path. Soc. of Lond. Sec. 45-58}\]

\[\text{Vol. 22. PP 360-384.}\]
Sweating and joint swelling, gangrene of the lower extremities, have also been described, this according to Soffroy and Achard to due to an obliteration of the arteries supplying the part, of alcohol nephritis. Helbigny also cites a case in which enormous thickening of the skin of the whole arm occurred, along with the formation of numerous perforating ulcers, making amputation necessary.

Mental alteration is, in these cases, if no means rare, and according to Tilting serves to distinguish alcoholic neuritis from other forms of neuritis; as Clark pointed out in 1883 it is not uncommon when we have the influence of lead and alcohol working together to have signs of psychical alteration produced and in this connection the point which Oliver successively that alcohol probably acts in solving the lead taken on his great hearing a loss of memory, sometimes amounting to actual amnesia (Korsakoff) and a general apathy.


Arch. de Med. Experiment. 1890 p. 129.


are the most prominent signs and have been frequently observed, moreover symptoms of a depressing character, or of suspicion or persecution, are occasionally to be noticed.

In addition to these changes described, one also, naturally finds frequently, the evidences of kidney, liver, jaundice diseases common to all cases of alcoholism.

Finally, Bjugard has observed in females, suppression of the menses during the course of this disease.

Mode of termination:

Some cases run a rapid course and terminate fatally in a few weeks or months; others end fatally after a year or two, this is the more usual cause. Many, provided that all for the alcoholic indulgence is stopped, recover in from 2 to 4 or more months. 2 Glainfelt's case in which pronounced atrophic paralysis and ataxy has developed, recovered in 8 months; such a case, however, is exceptionable, and in my opinion, when paralysis of both extremities, of the eye muscles and such like serious change supervene, recovery is rare. The cause of death is usually prostration, caused by debility or by paralysis of the Phrenic or vagus nerves leading to

cardiac or respiratory failure. Two forms of
Pott's disease supervene and both are
Pulmonary or extremity. Common in this form
of neuritis (Staddon, Tschayko et al.), in this connection
multiple the occasional occurrence of "Neuritis" in tubercular
subjects (Petits and Vaillard et al.) gives rise to the
hypothesis that the neuritis in cases of alcoholism
may be due to the bacillary influence, it is much
more likely however that the Phthisis is
in most cases a secondary result of degeneration
of the Vagus nerve.

Infective fatal toxine may be aided by
alcohol and kidney, due to Stomach disease, etc.

1. Loc. cit.

2. Loc. cit

3. Petits et Vaillard. Des neurites périphériques chez
les tuberculeux. Rev. d. méd. 86.
Pathology:

So many different views have been advanced by the legion of writers on this subject, with regard to the significance of the pathological changes observed in alcoholic polyneuritis, and concerning the nature of the process, its seat of origin and the direction of spread of the disease, that it renders a description difficult, and, by demonstrating a want of agreement among so many writers, proves that there must be still many links wanting in the chain which unite to complete the description of the disease. Leprince-Champion in 1868 gave the case of a smoker, in whom, at the autopsy, atrophy of the muscular spinal and peroneal nerves and early fatty degeneration of the muscles of the forearm was discovered, but it was not until as late as 1881 that the first clear pathological account of the degeneration which takes place in the nervous system was given by Dumesnix. There can therefore be no occasion for dissatisfaction in the progress that has been made. German writers, in this branch of the subject, unquestionably preeminate.

Gaz. hebdomat. 1881: "Dez paralysie alcoolique"
but Dance, represented by Dancer, Ducrey, Gombault and Peter and Vaillard, and England, by Breschfeld, Buzzerle, Hunter, Fray and Shankley, have also given valuable contributions.

Pathological Anatomy:

(a) In the Peripheral Nerves.

The peripheral nerves of the upper and lower extremities are in all cases found to be more or less degenerated, the degeneration varying with the length of duration and acuteness of the disease. The degeneration seems to be a 'pure paraneuritis' one, similar to the secondary degeneration which occurs in a nerve when it is cut off from its central connection, viz a segmentation and breaking down of the medullary sheath or white substance of Schwann, leading to a formation of granular collections and fatty droplets in the myelin, secondly, a swelling and proliferation of the nuclei under the neurilemma or 'sheath of Schwann', and then a destruction of the axis-cylinder, the products of this myelinic degeneration and the remains of the axis-cylinder thus disappear, and the neurilemma clumping in gives the appearance on transverse section of a wavy, modulated line of the nodes.
corresponding to the nerve nuclei), finally the place of the originally healthy nerve is occupied by fibro-cellular connective tissue. Shows this retro-parenchymatous regeneration be very acute and severe, an interstitial inflammatory change may occur in the perineurium and in the connective tissue between the individual nerve fibres, but in most cases this adventitial change is slight; further in all my cases, the nerve trunks when exposed at the post-mortem examination of the cadaver have presented nothing abnormal to the naked eye, which would not be the case were any extensive adventitial proliferation present. Nevertheless there are a few writers (Eichholtz, Hassen et al.) who lay stress on the importance of the connective tissue change, the latter even declares that the regeneration of the nerve elements is secondary to the adventitial proliferation. The regeneration is symmetrical and is greatest at the periphery, the smaller muscular limbs, especially, and the cutaneous branches show the highest degree of regeneration often only one or two healthy nerve fibres being discernible in them; proceeding centripetally, there is a gradual diminution in the number of fibres.

1. Eichholtz. loc. cit. p. 239.
In tre, and on examining the nerve trunks near their origin from the cord little or no segmentation is to be found in them, that is to say in cases which are not very far advanced. Certain nerves appear to partake in the degeneration to a greater extent than others; these are the anterior tibialis and the peroneal nerves in the lower extremities, and the radial nerves in the upper extremity. I cannot confirm Lorenz's observation that the anterior crural nerve enjoys a comparative immunity from degeneration. Eichhorst describes a form of inflammatory histological change in the muscle twigs, which he has specially termed "Neuritis Fascialis," that consists in an increase of the perineurium in the form of lamellae like the coats of an onion round the degenerated nerve, and the connective tissue envelops to such an extent as to form round and isolate muscles adjacent to the nerve. This change I have met with in cases of alcoholic neuritis but I have also seen it, in cases the nerve degeneration in perfectly healthy muscles. "Neuritis Fascialis" also describes that it is not pathological. The histological nerves have been found in cases (Pal). The Vagus 1, 2, 3, 4.


2 Eichhorst loc. cit.


nerves have already been observed by many to show the characteristic peripherally increasing degeneration (Dejerine, Chauveau, Pal, Stargardt) and so the diaphragmatic paralyses, the tachycardia and possibly the frequency of pulmonary tuberculosis in these subjects are accounted for.

With regard to the Cranial Nerves, I can find no mention in the literature of cases in which they have been to any extent degenerated, even when there has been in the same case, paralysis of the muscles supplied by one or more of these nerves, for instance in one of Chauveau's cases, in which clinically Ptosis and Abducens paralysis existed, the Oculo-motor and Abducens nerves were found on microscopic examination to be perfectly healthy, the nerve of one of my cases, showing similar clinical phenomena of examined with like negative result. In the ophth nerve, however, W. Hoff, has described the occasional occurrence of a

Dejerine, Contribution à l'étude de la rév. de l'encephalopie.
Arch. J. Phys. 20, 1st part. 1887.

Chauveau, loc. cit.
P. Pal, loc. cit.

S. Stargardt, loc. cit.

pronounced retrobulbar neuritis, this coincides
with the blurring of the Optic Disc, which is
sometimes to be seen ophthalmoscopically
during life.
No cases where the Sympathetic System has
been diseased have been described, but Procco
and Fusari, in many cases of Acute Multiple
Neuritis, mention affection of the Cardiac Plexus
and the Coccygeal Plexus, which they hold to
be the cause of the crises in that affection.

Change in the Spinal Cord and Nerve Roots:

The Spinal Cord has, by most
of writers, been described as absolutely intact or only
the seat of slight and unimportant change,
such as vacuolation and exuberant pigmentation
in some of the large ganglionic cells in the gray
substance, more particularly in the anterior
horn (Schulz, Dorenz etc.). With regard to
the vacuolation which occurs in the cells of
the anterior horn in this disease, most writers
regard it as of little importance and Schulz
not only declares that the change is not

1. Procco e Fusari: Una lezione contribuzione allo studio
clinico e anatomico patologico della neurite multiforme.

2. Schulz, loc. cit.

3. Dorenz, loc. cit.
pathological but that it can also be produced artificially. 3. Charcot supports this view; again, Kahler and Pick and 2. Leyden also state that these slight changes in the ganglion cells of the cord cannot account for the degeneration present in the muscle and nerves. 3. Eisenlohr, on the other hand, has met with extensive vacuolation in the cells of the anterior horn and declares that it is not only pathological, but a cause of the peripheral degenerative changes. Eisenlohr is to some extent supported by Strümpell; the observations of 4. Bower Lewis also tend to prove that the vacuolation metamorphosis in this relation is not so unimportant as it is stated to be, that observer has repeatedly found extensive vacuolation changes in the small cells of the 2nd cortical layer in the motor region in cases of Epilepsy and Chronic Alcoholic Intoxication, and in the

In the case he states, that this degeneration plays a most important pathogenetic role, the fact that all his sections were made from the fresh brain with the aid of the freezing microtome also disturbs the view that they are the product of imperfect methods of hardening etc., the same writer describes the change as being, primarily, the formation of a bright refractile globule of fat within the nucleus, which at a later stage breaks out and leaves a round or irregular shaped cavity. With respect to the more important changes that have been described, Schaffer cites a case in which the large cells in the anterior cornu, particularly in the lumbar region, were atrophied. In one of Hark's cases, slight inflammatory change were found in the cord, as well as in the brain and medulla oblongata, the changes in the cord were most pronounced in the lumbar region and least in the dorsal, the number of nuclei in the grey matter was increased, some of the vessels being full of them. The cells in the anterior cornu appear swollen, opaque, shapeless and deprived of their normal number of processes, and their nuclei had lost their susceptibility to staining.

In the case in which he observed leptomenigeal and degeneration of the posterior columns and anterior nerve roots, the posterior columns being especially affected in the peripheral border.

Oppenheim also refers to a case in which a similar patch was found in the cord in the upper lumbar region, which had caused atrophy of the right anterior horn and partial degeneration of the anterior columns.

Verost describes a remarkable case of a prominent descending degeneration of Goll's columns in a rabble. This case, though extremely interesting, does not present the typical clinical symptoms of alcoholic neuritis, and in addition the sciotic and radial nerves (apparently the only ones examined) were found normal.

In the spinal cords which I have examined, the pathological changes have always been considerable and in successive cases I found changes of the shape of scattered degenerated fibres throughout the white matter of the cord, with special involvement of certain columns, namely Goll's columns (min...
especially in the peripheral part, Leriche's tract, the anterior pyramids and of the lateral columns, the Lateral Columnar, these changes were most marked in the lumbo-sacral and lower cervical region; another case which was, however, probably a combination of Caves disease and alcoholic neuritis showed destruction of the Pons, external, Goll's, Leriche's and Clarke's columns as in a typical case of Caves.

After going through the literature, I can only find 2 cases, both reported by Pen, in which the cord was affected as in the three first mentioned cases, in Pen's first case, a similar degeneration was found in Leriche's tract in the lumbar region and in Goll's column in the cervical region, at the same time thinning of the commissures existed; in the second case, that of a man with a kyphotic spine, he found slight degenerative changes in the posterior columns in the kyphotic region, at the level of the cervical enlargement he found a slight amount of degeneration in Goll's column, and in the Lateral Columnar tract and at the level of the second and third cervical nerves in intense degeneration in Goll's column, and the peripheral part of the Anterior Pyramidal Tracts.

In two of my cases, I found a vacuolisation of the large ganglion cells in the anterior cornua in the cervical region; in several, excessive pigmentation of the large ganglion cells was markedly in the lower parts of the cord (but to this I attribute no importance) and in one case a slight degree of lepto meningitis.

Coming next to the anterior and posterior spinal nerve roots, the majority of observers describe them as being completely unaffected (Strümpell, Hallett, Aran, Eichholtz et al.); on the other hand, in Wilkins' and in Vierordt's cases, the posterior roots were degenerated and in one of Pol's cases both anterior and posterior roots were diseased. In nearly all my cases, I have found distinct disease of the posterior roots and in some cases affection of the anterior roots, in all cases the posterior roots were affected more than the anterior ones and in the whole I found more degeneration of the root fibres in the lumbosacral and cervical regions than in any other segment.

From these observations I am led to believe that degeneration of the root fibres is much more common than is generally imagined.

Changes in the Ganglia or the Posterior Spinal nerves have, so far as I know, only once
been described, namely by Auenbrugger, and in the hands of most observers they seem to have escaped observation altogether. In two of my cases I have found in the ganglia from the lumbar and lower cervical segments of the cord, degeneration of the nerve fibre running through the ganglia themselves and a comparatively high degree of degeneration in the root fibres running centripetally from the ganglia; the fibre on the peripheral side of the ganglia and in the anterior nerve roots, was comparatively healthy. Auerbrugger's observations were of a similar nature.

In the Medulla Oblongata and Pons Auerbrugger is the only writer who has hitherto described any pathological change; in one of his cases the glossopharyngeal nucleus was found to be highly degenerated, many of the nuclear cells had disappeared, the ground tissue of the nucleus was rarefied, homogeneus patches with a vessel in the centre were to be seen and numerous blood extravasations and a great number of large and small blood vessels were present; further the grey matter of the Acustico-spinous, extending from the Acustico nucleus up to the ponticus commissura, was studded with haemorrhages which

Auerbrugger, L. E. et al.
were most numerous in the neighbourhood of the Oculo-motoria nucleus, the 3rd nucleus of the 3rd and 4th nerves were themselves healthy. In this case it is interesting to note that clinically, Abscess paralytic, Platy, Hypoesthesia and Coëstalgia were observed, I would not like to affirm, however, that the condition described was essentially a product of Alcoholicism.

I have also observed scattered degenerated fibres in the Pyramidal fibre, in the Medulla Oblongata and Pons in cases in which at the same time there existed degeneration in the antenor pyramidal and other fibres of the spinal cord, but in no case where the Vagus nerves have been diseased have I found any appreciable change in the preceding nucleus.

Though I believe that cortical degeneration will be found when carefully looked for, still, it has only once been described, namely by those who found the ganglion cells in different parts of the cortex diseased. Change in the muscles of the muscles have been for any length of time paralysed, they will appear atrophied to the naked eye, and as a rule there which were first paralysed are the ones which show the most wasting, there, as already

1. [Footnote: loc. cit.]
mentioned, as generally the extensors of the foot, the Peroneus, and the extensors on the back of the forearm, three muscles show prominently the change which I am about to detail. At the muscles of the calf, the adductor muscles of the thigh, the Biceps Brachii and Deltoïd, the intercostal muscles (Pab) the Diaphragm (Thorax and Pab) and in fact any muscle in the supplying nerve of which degeneration has been discovered, may show signs of disease, and I may say, that as a rule, the pathological alteration which the muscle undergoes, varies with the amount of disease in the nerve or nerves, which supply it. Microscopically we notice an atrophy of and a loss of the normal striation in many of the fibres, these, also, when stained with basic acid often present a peculiar granular appearance, due probably to the existence of scattered particles of fat in the muscle fibres, in nearly all cases there is a strong proliferation of the muscle nuclei and this has been observed to take place to such an extent, that the spaces left vacant by atrophic fibres have been entirely filled up with nuclei (Hasten) ; others mention the occurrence of a faulty proliferation between the muscle fibres, but this appearance I have not been able to verify.
"Something recently found in one case, sign of such an extensive acute inflammatory condition, as to lead him to formulate the theory that the whole disease must be really in a primary myopathy. The changes in the muscles produced by Eichhorn, 'Neuritis Fascias', have already been mentioned. When the vague is dissected the heart muscle may also show degenerative changes, in this connection 3 Bejerne has recorded a fatty degeneration of some of the heart muscle fibres, 2 Sharkey has found vascular inflammatory change, and I have noted 'cloudy swelling' and 'brown atrophy' of some fibres.

Changes in the Blood vessels. -

Vascular changes in this disease have not as yet attracted much attention, and those who have written on the subject, appear to me to make too much of the pathological importance of the changes. 4 Munkowski was the first to describe a case of multiple neuritis with vascular changes, in this case he attributes the nerve destruction to the vascular change.

2. Sharkey loc. cit.
"Seffroy and Ackermann also describe a case in which there was thickening of the vessel wall with ulceration of the lumen, these changes were especially marked in the sciatic nerve, and in the left side, between the buttock and the lower third of the thigh no vessels were found which were not diseased; they also attribute the nerve degeneration to this vascular alteration.

Lorenz gives a very clear account of the vessel changes which he found in the case of a "potato" with multiple neuritis, in addition to the less important changes, all the peripheral arteries smaller than 1.5 millimeters in diameter but not less than 0.4 mm were diseased, a thickening of the intima and a partial cellular proliferation of the walls were the chief change, and had in some cases caused complete obliteration of the vessel, the vessels under 0.4 millimeters in diameter were curiously enough normal. Other writers (Russmann and Maser, Leyden, Vernier, Thomsen, Harkey, etc.)

2. Lorenz. loc. cit.
have also described changes.
I have also noticed changes in the vessels in the peripheral nerves and muscles which I have examined, such as thickening of the intima and adventitia of arterioles, congestion of the venules sometimes an actual organizing thrombus; or small haemorrhages, many of the smaller vessels, however, which I have carefully examined, in advanced cases have shown no perceptible change, and in any case, I should not care to declare that the vascular alteration is not produced by the primary nervous degeneration.
Pathology:

Here are some chief views concerning the pathogenesis of alcoholic neuritis:
1. That it is a purely peripheral degeneration, primarily in character and probably due to the direct action of alcohol on the peripheral nervous system.
2. That it is primarily myopathic.
3. That it is induced by vascular changes.
4. That it is the result of central degenerative influences.

If one accepts the view that peripheral alcoholic degeneration is a primary degeneration of the peripheral nerves, one finds oneself at once in conflict with the well-known Wallerian theory of degeneration which has stood since the year 1849, which, shortly put, is as follows:

a. That peripheral nerves only degenerate in the same direction as the impressions run which they conduct, that is in the direction of physiological conduct.

b. That this degeneration can only occur when the nerve is cut off from its trophic centre, or that centre is diseased.

Now, since the large cells in the anterior column of the spinal cord are generally admitted to be the trophic centres for, at any rate, the motor nerves, we should expect to find these cells...
Sick cells in all such cases, and these cells, which, in edema, have been found to be degenerate by several writers (Schulz, Living, Eisenlohr, etc.), but the changes have in nearly all cases been of slight importance, and only one of these writers, namely Eisenlohr, attributes any importance to the conditions found (Eisenlohr states that the vacuolation of the cells is the cause of the peripheral degeneration).

Having regard to these conditions, Ed. came forward with the view that the change in the cells of the cord might be a purely functional one, and so cause a peripheral degeneration without exhibiting macroscopic signs; in opposition to this hypothesis is the fact that areas (Strumpell's olives), why in these cases, are the anterior nerve roots not diseased, and how can the early symptoms of the disease, indicating disease of the peripheral sensory nerves, be accounted for? Ed's reply was, that the peripheral nerves being the furthest removed from the toxic influence, were, on account of their circulatory relation, the first to suffer.

Bogard, not long after this, advanced the theory that degeneration is due to a resistance on the vascular centres on the Medulla and the Spinal Cord.
The two French observers, Joffroy and Acher, and others, are inclined to regard a change in the smaller vessels as the primary cause of the regeneration, the alcohol circulating in the vessels producing an inflammatory effect on the vessel wall.

Hasner and others hold that the change in the nerves is chiefly an inflammatory pathological one, the fibro-cellular proliferation thereby set up, causes their regeneration, but as regards this view in the first place it is certainly quite explainable for a high degree of inflammatory pathological change to take place in alcoholic degeneration of the nerves, and secondly, even if it does occur (that is to say, primarily), it is still doubtful whether by pressure it would cause regeneration of the nerves, for I have seen in cases of Alcoholic Poisoning, an excessive proliferation of perineurium and endoneurium, the latter having grown round and widely separated the individual nerve fibres from one another, and yet the majority of these nerve fibres which were, so to speak, isolated, were healthy and were certainly not suffering from the effects of pressure.

Sternberg and Senat have had cases of Multiple Neuritis in which a pronounced inflammatory changes in the muscles with excessive nuclear
proliferation were the main pathological features of this, as already mentioned, has lead to the proposition of Sienert's theory that the disease is primarily a myopathy.

My own observations make me inclined to the view that the change is not a purely peripheral one, but that the toxic action of alcohol also works with considerable effect upon the Medulla Spinalis and also probably upon the brain, the psychical phenomena so often observed (Korenoff, etc.) possibly depend upon some central change such as that in the fifth layer of motor cells in the cortex, described by B. D'Anjou in his chapter on the Pathology of Chronic Alcoholism. Is it not the change in the Spinal Cord account for the failure of the Patellar reflexes, the bladder and rectal troubles, and the ataxy so often met with?

The number of instances in which the Spinal Cord has been found diseased is now considerable, and Pat's cases in which the Cord were examined by the most recent methods can be well compared with mine. The fact that degenerated fibres were found in so many columns not in connection physiologically is puzzling, and it is also difficult to state in which direction the disease
is travelling, ground, nevertheless, as
furthermore for assuming that the disease can
advance in a centripetal direction, since Virchow
and Strümpell cite cases in which degeneration
occurred against the conducting stream; on the
other hand it is not to my mind clearly proved
that the source of evil lies more centrally than
the Medulla oblongata.
Cases.


On account of general debility and loss of memory, the history was difficult to obtain. The mother is said to have died of Phthisis and 6 brothers and sisters are dead. He had been ill for 8 months and had spent part of the time in bed. Complaints of cough, pain in the chest, inability to walk, languor and loss of power in the legs. Eighteen months ago seems to have had similar symptoms but by temperance giving up his drunken habit he succeeded in freeing himself from them. Has for several years been a heavy schnapps drinker.

Status Praesens:

The face is pale, the normal paracentric boundaries.

The lungs show evidence of extensive tubercular disease. Respiration 36 per minute.

Examination of the heart reveals no abnormality. Urine also normal.

Nervous System:

No right side Placi and abscess.

Paralysis also bilateral nystagmus.

Sensibility and sense of localization of the skin.
of the lower extremity markedly reduced. Active and passive movements painful. No complete paralysis to be recognized and still the patient cannot walk. Patellar and Plantar reflexes absent. Brach reflexes feeble.

Movement of the arm ataxic.

Electrical examination:

No remarkable changes in the upper extremity. The extensor Femoris and Peronei muscles do not respond to either direct or indirect Faradic stimulation. The Flexor muscles contract with 32 m.m. Faradic and 0.5 M.A. Salvarsan. The Peronei show slow, wavy contractions on direct Salvarsan stimulation. The electrical examination otherwise were normal.

Clinical Diagnosis

Alcoholism. Neuralgia.

Tuberculosis Pulmonum.

Arthritis Hepatitis.

20th August. The muscles supplied by the Radial nerve and the Cubical Anterior muscles paralyzed & extremely painful to pressure.

1st September. Death from Shock here.

Autopsy performed in the Pathological Institute 16 hours after death.
Post Mortem Examination.

Beneath each covering an area of about 7 sq. cm. centimeter in both trochanters and on the sacrum. On both knees and heels the skin is reddened and the subcutaneous tissue swollen.

Scalp normal. Calvarium of normal density. Dura Mater not stretched, all the venous sinuses filled with dark clotted blood. The Internal Meninges unusually bloody on the convexity, congested and thickened together. The Parietal can be separated in large lamellae from the cortex; Subarachnoïd fluid not excessive. The Brain itself of normal shape, the convolutions of the same obviously atrophied in the frontal region; the Sulci wide and shallow. The white substance soft, pale and glistening on section. The lateral ventricles somewhat dilated with fluid. The basal ganglia small. Some perivascular lymph spaces dilated. Right lung adherent at the apex; contains numerous Caseous granules masses throughout its substance but these are in greater numbers at the apex. Left lung also adherent to chest wall by vesicles at the apex; the surface of the lower lobe to covered with fresh fibrinous exudate, under which numerous grey military nodules.
are to be seen. There is no pleuritic exudation.
On section the upper lobes appear softer with caseous nodules the size of a cherry stone and also shows widespread caseous infiltration.
The liver to be is sufficiently consolidated and presents here and there milky nodules.
The liver is enlarged but otherwise it and the remaining thoracic, abdominal to other organs show nothing remarkable.

The spinal cord in the fresh condition is soft and pale.

A dissection of the muscles of the liver and upper extremities shows especial weakness of the 3 Peroneal muscles and the Tibial anterior up to the 5th toe in the leg, one of the Extensors of the wrist, the Extensor Communis Digiti, and the Extensor Minimi Digit in the forearm; on section these muscles are all of a septic color.

Microscopical Examination:

The Pons, Medulla Oblongata, Medulla Spinalis, portions of various nerves and muscles were hardened in Müller’s fluid and alcohol, immersed and cut in albumin and stained (1) according to Weigert’s method (some also were stained after Pali’s modification of the same) (2) with Bie’s Alum Tintinental.
The muscles were also stained with ordinary
Haematoylin and cosine. The cord through being somewhat decomposed when removed from the cadaver, only admitted of thin, serviceable sections being made in the upper cervical, lumbar and sacral regions.

Pro and Medulla Oblongata:

A series of sections was made from the crossing of the Pyramids to the posterior root of the spinal cord, and every tenth section examined, nothing pathological was revealed beyond a thickening of the pyramids of the ventriculus quartio and the existence of a few degenerated nerve fibres throughout the Pyramids.

Medulla Spinalis.

The sections at the level of origin of the 3rd lumbar pair of nerves show disorganized degenerated fibres throughout the white substance, a few disorganized fibres in the anterior and posterior roots, and a cellular obliteration of the central canal.

The sections from the levels of the 5th lumbar and 3rd sacral pairs show the same condition somewhat more marked as regards the white columns, and a deeper involvement of the posterior roots.

Peripheral Nerves:

Medianus (Dexter). Considerably degenerated, much fibro-cellular tissue with numerous nuclei, and diseased nerves in all stages of degeneration.
Nervus Tibialis Anticus (Dexter)
(Cochineal Alum.)
× 35.

Showing that there is no marked increase of the Perineurium.

Nervus Tibialis Anticus (Dexter)
(Weigert)
× 150.

The healthy nerve fibres stand out black.
The diseased nerve, cellular and fibro-cellular elements remain unstained.
Nervus Ischiadicus (Sinister)
(Alum Cochineal)
\( x \, 400 \)

Fibre devoid of its contents, represented only by neurilemma and nuclei.

Healthy fibre

Fatty Body

Diseased fibre

Nucleus

Same Nerve cut longitudinally.
(Weigert.)
\( x \, 500 \)
to be seen. The degeneration is parenchymatous and in a rough estimate about 30 per cent of the fibres are diseased.

Ulnaris (Dexter) shows a somewhat lower degree of degeneration than the Median. Radialis (Dexter) is more affected than the Ulnar.

Many of the capillary vessels among the nerve fibres show thickened walls and in parts are thrombosed. Ipsiadiceus (Sinius Dexter) is less degenerated than the Median nerve.

Extensor Carpi Ulnaris (Dexter) more degenerated than any yet mentioned, but few healthy nerve fibres visible. The blood vessels in these nerves similar to those in the upper ext. 9.

Muscles:

Sustancia

Communis Digitum (Dexter).

Some fibres are atrophic and in such the nuclei are increased in number, most are normal and have not lost their annular marking.

Extensor Communis Digitum (Dexter), many more fibres are atrophic and show nuclear increase, sections of nerve bundles are seen to contain only one or two healthy fibres and the perineurium is thickened.

Extensor Carpi Ulnaris (Dexter) similar to the last mentioned muscle. Most of the vessels are
normal but some have thickened spinal & adventitial.

Anatomical Pathological Diagnosis:
Neuritis Alcohólica.
Tuberculosis Chronic Pulmonum.
Pleuritis Tuberculosa.
Atrophia Cerebri (grábus levióris).

Remarks.

This may be taken as a fairly typical case of alcoholic neuritis. The Paresis and Adductor paralysis are remarkable in so far as no change was found in the S. N. H. nerves to account for them.

The electrical changes correspond fairly to the nerve degeneration revealed.

The Atrophia Cerebri is also of importance and I regret exceedingly that no examination of the cortex was made.
Case 2.

Clinical Report:

Name: A. S.
Occupation: Bookkeeper
Age: 34

A Bohemian living in Prague, married.
Admitted to the General Hospital, Prague, Sept. 10th.
Died: October 23rd, 1888.

History: Family history good. Has malarial fever 12 years ago when in Russia. Two years ago was divorced from his wife and from that date has led an immoral and intemperate life. A year ago was treated in the General Hospital in Prague for disease of the liver extreme. A month later he came again to the Hospital with ulcers in the feet (diagnosed as Ecthyma). For six weeks he has complained of general muscular weakness, cramps, and pains in the calves.

Present Condition (Sept. 14th, 1888):

of moderate size and strong build, muscularly powerful; has the general appearance of a Turk.art.

Alimentary System:

Tongue moist and purulent, also markedly tremulous. Liver much enlarged, can be palpated 2 inches below the costal arch on the right side, it is painful on pressure.
Spleen also enlarged reaching from the 7th rib to a point 2 fingers breadth below the costal arch, when it can be palpated.

Respiratory System: Comminucy Phthisis Pulmonalis was diagnosed. Respiration 38 per minute.

Circulatory System: Heart rapid, but regular. Apex beat can be only indistinctly felt.

Pulse 104, rhythm, and full.

Urine normal. Lower extremities and Scrotum swollen.

Nervous System:

a. Sensory Functions.

In the lower extremities patches several centimetres in diameter can be found, where the cutaneous sensibility is impaired; a delicate needle prick is not felt, but a deep prick is felt; very and correctly localized; the rate of conduction of a painful sensation is prolonged. Sensibility to heat and cold are unchanged. The muscular sensae is distinctly impaired. Papil reactions normally to light and accommodation.

b. Motor Functions.

Organic reflexes normal.

Skin reflexes exaggerated. Cerebral reflexes completely absent. Movements of the arm, undisturbed and normal意向 cannot stand on his leg.
Excitability of Paradoxic Stream.

Upper Extremity

Diploe. 90 90
Suprains. Diploe. 0 0
Ext. Dig. Comn. 65 65
Flex. Dig. Comn. 70 70
Flex. Subl. Dig. 70 70

Lower Extremity

M. Peroneus. 0 0
Abd. Anticus. 0 70
Ext. Dig. Comnus. 0 0
M. Peron. Long. 0 70
Gastrocnemius. 34 74

Examination

The patient cannot walk nor stand alone. The hand is slightly bent at the wrist, and the fingers at the metacarpal and phalangeal joint. The fingers of the thumb and first finger less so than the rest. The hands are tremulous. The fist cannot be completely closed voluntarily, and to the hand is with difficulty extended at the wrist. Other movements are fairly normal.

Both lower extremities are flexed at the knee, only slight voluntary movements can be performed without pain, but passive movements to a normal extent. Both feet are pointed at the ankle. The muscles of the upper extremity of normal volume, the lower extremities being estimators, it is impossible...
to state what degree of atrophy is present. Mentally the patient is dull and stupid and has been delusional e.g. he imagines silver coins are lying under his skin, which he can produce at will.

Clinical Diagnosis:
Neuritic Alcoholism Multiplex.
Tuberc. Pulmonum.

18th June.
Oct. 22d. No abnormal sensations of touch.
26th. Paralysis of left arm worse, also leg cannot raise the legs in bed. Tense at pain in pressure in calves of legs.
Nov. 1st. Shrimp sign of exhaustion generally. Desatellite on shoulder blade and sacrum. Pulse very rapid 46.
20th. Has developed a Pneumonia of left lung. Sleeps badly in account of pain in the left. Cannot rise raise his arm.
Temperature 103.
23rd. Died suddenly.

P. N. Exam. 15 hours after death in the Pathology Institute.
For Report (over).
Nothing to learnt regarding external appearance.

Head:

Scalp pale, calvarium measuring 32 cm. in its horizontal circumference, dense and thick. Dura somewhat stretched, its sinuses containing a small quantity of dark clot. Internal meninges thickened, hyperemic and abnormally adherent to the cortex. Basal cistern normal.

Brain small, frontal convolutions appear somewhat thinned, brain substance soft, vessels not congested. Cerebral ventricles not dilated; Basal nerve apparently healthy.

Lungs:

Pulmonary pleura of both upper lobes covered with numerous confluent ecchymoses and grey miliary nodules. In the lung substance are masses of the size of walnuts, formed by the aggregate of miliary nodules, hairs and thorns to be found.

Heart muscle brown and tender.

Liver enlarged, capsule smooth; on section pale yellow in colour and friable.

Spleen enlarged, thick and in the kidneys miliary nodules discernible.

Pancreas shows a peculiar cystic degeneration.

The spinal cord and nerves showed nothing pathological to the naked eye.

The rest of the oblongata & diencephalic and spinal cord were
hardened in Müller's fluid and alcohol, mounted in Celloidin and stained with Alum Cauimal and according to Weigert's method. The nerves were further flattened and stained with Acetic Acid according to *Marchi's method, and also with Alum Cauimal and Weigert's membranes.

MCulla Olingua and Pons.

Sections were made at the level of all the nuclei situated between the decusation of the Pyramids and the posterior Corpus Quadrijugia. These nuclei along with their nerve roots were in all cases found to be healthy. Degenerate fibers to be seen scattered throughout the Pyramids. Sections from the spinal cord were taken from


1) Sections cut as small as possible are placed for 8 days (better longer) in Müller's fluid.

2) Left for 8 days in a mixture of
   1. Müller's Fluid 2 parts
   2. Acetic Acid 10% 1 part

3) Washed very thoroughly in water.

4) After hardened in Alcohol of increasing strength.

5) Imbed in Celloidin.

The degenerated fibers appear black, the rest light gray. After staining in Carmen is possible, and also Weigert's method, provided the preparations have been long enough in Müller's fluid.
Anterior Pyramids.
The dots indicate the degree of degenerations found. To also see the drawings of the Spinal Cord.

6th Cervical.

3rd Dorsal.

12th Dorsal.

5th Lumbar.

3rd Sacral.
the following levels: 3rd and 6th cervical nerve roots, 8th and 12th dorsal roots, 2nd lumbar, and 1st sacral.

In the cervical region, degenerated fibres can be seen scattered throughout all the white columns, most however are to be found in the anterior pyramids (periphery apart). Lesions of these zones and the periphery of the posterior columns. A few cells in the anterior horn are vacuolated and others are more deeply pigmented than normal. The Pia Matar in this region is thickened. The anterior and posterior roots to the contain degenerated fibres.

In the dorsal region, the nerve roots are again seen to be degenerated and at the lower level the 1st root is more degenerated than the anterior. The same condition exists in the white columns as in the cervical region, but here, more numerous. Clarke's root column and the cells in the anterior and posterior horns appear healthy. The central canal is obliterated by a proliferation of its epithelium.

In the lumbar region, the posterior nerve roots are rather highly degenerated, the anterior hardly at all. In the white columns in the region few degenerated fibres are seen anteriorly, but the degree of degeneration posteriorly is greater, the posterior root zone being especially affected.
The black stained fibers are degenerated, the others healthy. Unstained fibro-cellular tissue lies between the fibers.

Nervus Phrenicus Dexter.
(Marchi) x 150.

Blood Vessel. The healthy nerves stand out prominently; the faintly stained tissue consists of the remains of degenerated nerve and fibro-cellular tissue.

Anterior Roots of the 6th Cervical pair of nerves.
(Weigert) x

Posterior Roots of same.
(Weigert) x 250.
Posterior Root highly diseased.

Anterior root showing scarcely any regeneration.

Ganglion on the posterior root of the 5th right lumbar nerve.

Hecht. x 20.

Nervus Radialis (dexter)

(Weigert)

x 550.
Some of the nerve cells are deeply pigmented but otherwise present no abnormality. The central canal is obliterated.

In the sacral region the posterior roots are more degenerated than the anterior not at all.

Other nerve cell appearances are the same as those in the lumbar section.

The vessels of the cord appear healthy.

An examination of two posterior root ganglia from the 4th lumbar and 6th cervical nerves shows regeneration of comparable size fibres in the dorsal root of the ganglia, of few also in the Vagus (Dext.) ganglia. 

Conversely, considerably degenerated fibres were in all stages of degeneration and also a marked nuclear proliferation visible between the fibres.

Vagus (Venis) somewhat less diseased than the right.

Phrenicus (Dext.) and (Venis) also seat of an acute destructive process, a considerable mitotic like proliferation in the sheath small haemorrhage have here as there occurred.

Medianner (Dext.) contains few healthy fibres and also few fibres in a process of degeneration to be seen due to pronounced fibre cellular proliferation. The sheath of the nerve is not thickened.
Musculus Peroneus (Sinister)  x 400.
(Wiegert's Hæmatoxylin.)

Nervus Vagus (Sinister)  (Marchi.)
 x 300

Same nerve cut longitudinally.  (Marchi)
 x 300
Radialis (Dexter)

Shows a more advanced stage of what is found in the median.

Lumbalis Quartus (cut within the Spinal Canal); also degenerate more in some fasciculi than in others.

Pernecus (Dexter)

Presents a more extreme degree of degeneration than any yet examined, almost the whole field being taken up by fibro-cellular tissue.

Pernecus (Sinistri)

Same as right.

Muscles Pernecus (Dexter & Sinistri), many of the fibres are shrunken and have lost their normal striation. The muscle nuclei are greatly increased in number. The nerve trunks present only one or two healthy fibres.

Anatome - Pathological Diagnosis:

Tuberculosis Chronicum Pulmonum.

Pleuritis tuberculosa Naturalis.

Neuritis Multiplex Alcoholica.

Necrosis Chorioides Chronic.

Staurosis Hepatis.

Globus Hæmorrhag. Pancreatis.
Remarks:

The most noteworthy point in this very interesting case is the existence of regeneration in the spinal cord and nerve roots. The more specific affection of the posterior white columns in the lower regions of the cord and of the anterior columns in the cervical region and of disease of the medullary tract throughout its course is remarkable; the condition of the nerve roots and of the spinal ganglia is also interesting.

Disease of the Phrenic & Vagi nerves accounts for the abnormal cardiac and respiratory phenomena observed clinically and also probably the sudden death.

The existence of Phthisis Pulmonalis, a mental aberration and vaso-motor change (echtyma and oedema) are also interesting in confirming the observations of others.
Case 3.
Name R. J.
Age 6½ years. Sex: male.
Admitted to the Children's Hospital, Pray under Professor G. K. Hufner on Jan. 6th, 1832.
Died Jan. 23rd, 1832.

History. No the signs of Phthisis Pulmonalis, a worthless an idiot. The child has always been weak and delicate and the parents imagining that alcohol would help to strengthen it has been for the last 2½ years in the habit of administering considerable quantities of wine and beer daily. About a month before coming it to the hospital the father noticed that the child's powers of progress were becoming enfeebled, mentally he also became duller and was frequently found crying. The child's legs gradually became weaker and now he is unable to walk at all.

State on admission:

The child is thin and anaemic but no special wasting of the muscle of the extremities is noticeable. It appears he is unable to walk; the leg can be bent at the knee without trouble. Dorsiflexion at the ankle is, however, impossible, the toes are pointed and the calf muscles slightly contracted. The hand grip is very feeble and
is stronger on the left side than on the right. The movements at the wrist are still impaired, those at the elbow and shoulder joint normal. Temperature on admission 99.4 F. (The temperature varies between 99 and 101 F. throughout the illness, with the exception of the last 2 or 3 days).

Appetite is fair and the child of ten ounces. Food, fluids, constipation. Circulatory & respiratory system apparently healthy. Urine contains a considerable amount of albumen and uric acid.

Sensitivity to touch on the back & of the feet impairs. Sensation for heat and cold normal. Myotonic irritability of the Peroneal and extensor muscles increased.

Sensory reflex and ankle clonus not to be elicited, the superficial reflexes normal.

The Perineal & Muscles. Pelvic nerves & the Perineal and calf muscle papilled on pressure. The Perineal muscles also shows diminished excitability to electrical stimulation.

The chief showed no signs of improvement & died on the 23rd of January from Pneumonia.

Clinical Diagnosis:

Neuritis.: Alcoholica Multiplex

Morbus Brighti.

Gastroitis. Acmea.
Post Mortem Examination

Body 110 cm long, weakly built, in the skin on the outer surface of the upper arm is an ecchymosis or ecchymotous eruption, the result of vaccination.

Scaphoid pale; calvarium measures 45 cm in its horizontal circumference and is of normal thickness; the suture lines contain fluid but soft clot. The internal meninges thin, not congested. Basal arteries normal.

The brain large, not congested but watery.

Right lung chiefs to the right side on account of a fibrinous exudation and the liver to be in its mediastinal surface is firmly adherent to the pericardium by a thin adhesion. There are a few patches of pneumatic consolidation in the lower lobe.

The left lung is completely and firmly adherent to the diaphragm wall and the liver also. The lungs contain a few luetic nodules in a condition of pneumatic reduction. The bronchi and bronchial tubes of both lungs contain mucous serous fluid.

The pericardial and visceral layers of the pericardial sac adherent to one another.

The liver is enlarged, 1050 grams in weight, tense and congested, showing an exquisitely nutmeg-like appearance. Capsule thickened.
Spleen also enlarged and firm - capsule thick.
Right kidney congenitally displaced,
occupying a position in front of the Promontory
of the Sacrum. The vessels, ureter, etc correspond-
ingly altered but the right Suprarenal capsule
is normal in position. Both kidneys are
large, firm and congested.
In the lower part of the Pneum and in the
Caecum are a few tubercular ulcers, 1cm in
Diameter and undergoing a process of
Acarinisation, in the corresponding Mesenteric
area a few enlarged tubercular glands.
The portions of muscle & nerves removed and
the Spinal Cord show no naked eye alteration.

Microscopic Examination:
Portions of the muscles, nerves
and Spinal Cord were hardened for Marchi
the method of staining and also for Wiegert's
method and for Alum Coehnicnt. The muscles
were also stained with Eosine and Haematoxylin.

Spinal Cord:
At the level of the 3rd Sacral nerve roots.
Show degeneration of both anterior and
Posterior nerve roots, the posterior being most
affected. Disseminated degenerate fibres as seen
throughout the white substance but they are
more numerous in the peripheral portion of the
nervous system. The anterior lateral containing
fibres are more numerous than the posterior lateral containing
fibres.
The dotted parts indicate where most diseased fibers were found.

3rd Sacral.

5th Lumbar.

12th Dorsal.

3rd Dorsal.

6th Cervical.

3rd Cervical.
tracts and Lissauer’s tracts. A few axonal bodies also to be seen. Large cells normal.
At the level of the 3rd Lumbar roots.
Somas Saccal, but the anterior roots are not too much affected.
At the levels of the 12th Dorsal pair -
both anterior and posterior roots are
again considerably diseased. Fibres in the
dorsal denting layer and direct to efferent
tract run affected.
At the level of the 3rd Dorsal pair
nerve roots less diseased - the posterior
columns also less affected - the anterior
Pyramid & anterior external columns contain
many degenerated fibres. Lissauer’s column still
highly degenerated.
At the level of the 6th Cervical pair
much less degeneration in the anterior
and posterior roots and also in the white
columns.
At the level of the 3rd pair.
the change are again less marked.
Peripheral Nerves:
Nervus Medians (Dexter)
Considerably degenerated, very
little interstitial proliferation and nuclear
increase.
Medians (Sinister)
some as Dexter.
remains of a degenerated nerve.
Neurilemma almost empty.
showing swollen axicylinder.

Spinal Cord.
Sixth Cervical.
Goll's Column.

(Alum Cochineal.)
× 400.

Muscular Tibialis Anticus.
(Cochineal Alum)
× 400.

To illustrate muscle atrophy, the increase of nuclei and Eichhorn's neurotus Fasciatus
in which the perineurium proliferates and forms round muscle fibers.)
In radialis (Dexter)

...and nuclei about 30 per cent. of the fibres are degenerated. Some of the arterioles have a markedly thickened media.

Tibialis Posticus (Dexter et Sinister)

Changes more advanced than in radialis.

Tibialis Anticus (Dexter et Sinister)

Degeneration still more marked.

Muscles:

Brachialis Anticus (Dexter et Sinister)

...a few fibres atrophied and presenting nuclear increase, most normal Derminal nerves hsz highly diseased, showing increase of Perineurium and Enchirring Neurilem Fascia.

Semitendinosus (Dexter et Sinister)

Fairly healthy (nerve hsz also not much degenerated).

Gastrocnemius (Dexter et Sinister)

...only a few atrophied fibres and not much nuclear increase. Some thickening of the vessels and neuritis fascians.

Remarks:

This case is of special interest on account of the young age of the patient and on account of the changes present in
the Medulla Spinalis; and it is further as far as I can gather, the youngest case on record in which the clinical signs of alcoholic neuritis have been confirmed by a Post Mortem Examination. Leszynsky reports clinically, a well marked case of multiple alcoholic neuritis affecting both the motor and sensory nerves in a child aged 7. foot and wrist drop were both present. The subsequent history of the case was not ascertained but it is known that the boy died in a few weeks.

The more or less severe affection of the spinal cord is so difficult to explain and its irregular distribution in the whole of the spine to the presumption that alcoholic neuritis is nothing more than a peripheral, commonly occurring disease no longer or its extension to the peripheral nerves.

The degeneration in the reversal of the white columns was of the typical character described by Legan, Rahn and Pick, and others as occurring in secondary degenerative affections, in place the space occupied formerly by a healthy fibre was filled by fibro-cellular tissue. Empty spaces representing the neurilemma devoid of its contents were to be seen, and this in two thin neurilemma in other

Leszynsky. Alcoholic Paralysis in a child.
cases contain granular and fatty debris or a very much swollen axi-cylinder.

The absence of marked sign of Pulmonary Tuberculosis is noteworthy.

The condition of the liver and kidneys can be partly attributed to the effect of alcoholic excess.
Case 4.
S. J.

Aged 37, a laborer, single.
Admitted to the Bray General Hospital January 28th 1882.
Died February 5th 1882.

Anamnesis:

Family history good. No previous illness, but for a year has had cough with yellow expectoration and pain in the chest and labored shortness of breath.

He has been a heavy drinker, taking 4 or 5 litres of beer daily for months. Signs of alcoholic paralysis occurred six weeks ago, in the form of pains of a rheumatic character in the muscles of the calf which were soon followed by paralytic signs in the legs, a few nights later the arm muscle became affected. For the past week has had oedema of the feet also loss of appetite, fever, night sweating and diarrhoea.

Status Praecox (Jan 25th 82)

Patient poorly developed, muscles small and flabby. Both feet and ankles oedematous.

Suffering from tuberculosis of intestines is very exhausted.

Heart's apex beat cannot be seen nor felt, its action can be heard to be rapid but the other sounds are in distinct in account of lung bronchial rales.

Pulse rapid and thready.

Dumpy jaw sign of advanced tuberculosis at both apices, but bronchiectatic rale are everywhere audible.
Integumentary, Urinary & Reproductive System present as being remarkable.

Nervous System:

With the exception of slight impairment of the sensibility to touch and pain in deep pressure in the nerves and muscles, the sensory functions are normal. He is suffering from retention of urine and needs catheterisation.

Patellar reflexes can be elicited on both sides, but are not active. No ankle clonus. Biceps reflex present. Skin reflexes normal. There are no ataxia signs.

Mentally he seems clear; sleep drowsy.

The feet remain plantar and dorsiflexion at the ankle joint is impossible, pressure on the extensor parts & on the Anterior Tibial, Extensor, Pronator & Calf muscles is especially painful. The action of the Quadriceps, Adductors & Ham Strings are little impaired. The muscles supplied by the Radial nerve are all weakened in action, but the other arm fairly healthy.

Six 3 days after admission from heart failure and asphyxia of the lungs.

Clinical Diagnosis

Cardiopathia Chronic Pulmonum.

Acetum Excre. Intemini.

Neuritis Alcoholici (gradus lesioni).
Pathological Report:

Beyond the vessels of the liver, extravasated blood was observed. The liver was not unusually enlarged.

Scalp: pale. Calvarium of normal thickness, 56 cm in its horizontal circumference. There was a streaked pattern on the scalp, containing dark blood.

Re: Throat: Slightly closed, not congested & easily detachable from the cortex. Brain apparently healthy.

On the vocal cords, in the right side, a tubular calculus of the size of a millet seed.

In the upper lobe of both lungs, numerous cavities are situated filled with greenish yellow pus.

Throughout the lung are numerous caseous masses, as large as a pea. Borders of lung emphysema.

Heart & large vessels apparently healthy.

Lung of normal size, its capsule granular, of firm consistence, pale brown, yellow color, vessels congested.

Kidneys of normal size, not with adherent capsule.

Muscle membrane of muscle thickened and covered with small hemorrhagic erosions.

An extremely advanced state of Emaciation United in the small intestines and about the spleen.

The mesocolon & tons were pale & yellow, as well as the intestines of various nerves & muscles, the latter and the special ones for examination according to Marchi method.
Result of Microscopical Examination:

Medulla and Pons:

In the left Vagus nucleus some of the nuclei of the cells are indistinct, in the nerve root also a few degenerated fibres can be seen in excess of connective tissue nuclei.

Otherwise as abnormal in these parts.

Spinal Cord:

Cervical: a few degenerated nerve fibres can be found in all the columns of the cord; the posterior nerve roots are also slightly diseased.

Dorsal: in the lower region one finds an increased no. of diseased fibres, espec. in Goll's column and Deiter's root zone. A few amphi.

bodies to be seen here and there in the periphery.

Cells in Clarke's column deeply pigmented.

Central Canal quite obliterated by epithelial growth. Some epithelial then thickening of the meninges and the adventitia.

Both the posterior and ant. roots are degenerated in the lower dorsal region.

Lumbar: much the same as the dorsal, but perhaps more degenerated fibres are to be found & the roots are more affected. The cells of the ant & posterior horns are deeply pigmented.

Sacral: as lumbars.

Nerves and Muscles (over).
Causa: Constans and Eponimus roman.

Medianus (Dext.) - only a slight deg. of degeneration about 10% of the fibers destroyed.

Mus. (Sinistra) in the same condition.

Schiadicus. (Dext.) a much higher degree of degeneration than in median with a disappearance of many more healthy fibers.

Schiadicus (Sinistra) same as right.

Popliteus (Dext.) shows a destruction of most fibers than the .

Piriformis (Dext.) highest degree of degeneration of all.

Muscles:

Quadriiceps Extensor (Sinistra)
Beiceps Brachii (Dext.)
Ac. Delt. Mag. (Sinistra)

All Piriformis (Sinistra) show slight nuclear proliferation.

Gemmellus super (S) shows nerve fiber as constantly degenerated.

Arthritic. Pathological Diagnosis:

Arteriosclerosis Chronicum Palmarum
Arteriosclerosis Longa et Extrema
Arteriosclerosis Hepatica (grad. levior)
Morbus Brighti Chronicus.
Neuropathia Alcohólica Multiplex (grad. levior)
Case 5.

J. S.

Admitted into the General Infirmary Prag, to Professor Plíhram's wards March 22nd 1832.
Died on the 1st of April 1832.

History:

His father and a sister died of some 'lung disease.' Two other sisters died in childhood.

For several years has indulged to excess in wine and beer. Has also suffered from gout and rheumatism. Patient had malarial fever in Wallachia in 1830.

His present illness began 3 months ago, patient first observing sensations of numbness and paresthesia in the feet, then in arms followed by weakness in the upper and lower extremities; he became unable to play his violin and soon became exhausted when walking. These locomotive troubles have continuously increased. Over week ago, he observed that his abdomen was swelling; the muscles he continues gradually up to date. In some time he has been troubled with cough, with purulent expectoration, cold of flesh & night sweats. Appetite has been good and the bowels regular.

One month ago the patient noticed a pronounced
mixture in the daily amount of urme that he passed and he has lately noticed a marked diminution in the same.

State of Abdomen:

of medium height, slim, with thin skin and naturally weak musculature.

Pancreas Deficient. Expression of face drawn and anxious, looks obtuse as well as paralytic.

Alimentary System:

The abdomen is much distended and measured 68 cm. at the navel, the superficial abdominal veins evidently distended. A considerable amount of fluid in the peritoneal cavity.

Liver markedly enlarged reaching in the right lobe to a point 8 cm. below the 12th rib on palpation firm with uneven surface.

Hematopoietic System:

Spleen also enlarged. Tumors in peritoneum obtained superiorly at 7th rib and extending downwards to a point 2 fingerbreadths below the 12th rib can be felt as a firm rounded body.

Circulatory System:

Apex beat cannot be seen or felt. Heart action weak and rapid - sounds healthy.

Pulse rapid, 140, weak, rhythmical.

Respiratory System:

Signs of consolidation at both apices. Rales to basilii present in large grains in the
Speculum (Examine by Uhrleko method).

Integumentary System:

- Lower extremities: not otherwise
- Same covered with numerous brownly pigmented spots
- The size of peas

Urine: ant and albumen - older reaction now.

Nervous System:

- Sensory Function:

  The sensation of numbness on the right
  horizon of both feet & on the left wrist, sensitivity to
  touch impaired in some parts of the leg and
  thigh, otherwise normal. Can distinguish heat and
  cold perfectly.
  Muscular sense markedly impaired.
  Fundus oculi normal, and pupil also. pup and also reach normal.

- Motor Functions:

  Patellar & Gracilis reflexes as well as
  the ankle clonus fail. Cremasteric reflex absent,
  but superficial reflexes normal.

Vasomotor Function:

  Perspiration exceedingly

Central Nervous Function:

  No fall and stature, attention difficult
  to command, memory markedly impaired, fits to
  his age (child was given by a point), speech
  thick and slow. No delusions. Sleep badly.

- Oculomotor System:

  Cannot walk alone; is unable to
  bend his neck on account of severe pain.
Les severe cramps especially during the night.
No rigidity or twitching or tremor.
All the muscles below the knee are distinctly wasted, those of the upper extremity appear of normal volume, but pressure on them as well as on those of the lower extremities causes pain; pressure on the trunk of the muscles--sacral, umbra, sciatic and femoral nerve trunk also occasion pain.

An electrical examination was not attempted.

Clinical Diagnosis:

Neuritis Multiplex Alcoholic
Anemia Hepatitis.
Cancer Liver Chronic.
Acute Tumour.
Tuberculous Chronic.
Nephritis Chronic.

The heart's action becoming feeble and more rapid and congestion of the lungs supervening, caused the patient's death about a week after admission.

The autopsy was made the day after death at the Pathological Institute with the following

No remarkable external appearances.
Calcium moto aphaelic, measuring 5-3/4 cc. with horizontal circumference of normal shape.
Dura, peri & durae apparently healthy.
In the lungs, numerous grey oily nodules with the staining of the parenchyma in both upper lobes, grey nodules, the size of a pin's head also spread throughout the lung.

Heart: in the endocardium of the right & left sides scattered milky nodules; in the right auricle a large yellow tubercular nodule attached to the wall and half filling the cavity.

Peritoneal sac contains 3 litres of yellowish cloudy fluid.

Liver, amyloid and cirrhosis.

Stomach shows signs of chronic gastritis.

No changes in kidneys but a few white nodules in the pelvis of the

Kidneys enlarged, capsule non-adherent, cortex irregular, in the same numerous milky nodules.

Parenchyma firm.

Suprarenal capsules both enlarged and caseated.

The spinal cord, sinus with myel macrscopic, hardened and stained according to Marchi's method it shows no myelin substance; degeneration in the posterior roots and posterior columns in the Sacral, Lumbar and Dorsal regions.

Notes:-

Portions of the following were cut out and stained with osmic acid, after fixative method, and examined microscopically.
Vagus, Phrenicus, Medianus, Ulnaris, Ischiadicus,
Cruralis anterior from both sides.

The Vagi both show a considerable of degenerated
fibres and nuclear proliferation.
Adventitial changes slight.

Phrenicus affected similarly to the Vagi.
Medianus: only a slight degree of degeneration
Ulnaris: also not much degeneration but more
than the Medianus.

Ischiadicus: shows comparatively large number of
Degenerated Fibres.
Cruralis anterior: few degenerated fibres.

Many arterioles and venules in these nerves
have intima and media to very much thickened.
A portion of heart muscle taken from the left
ventricle shows many atrophies, pigmentation and
granular fibres.

Anatomico-pathological Diagnosis.
1. Tuberculosis Chronic Bovorum et
   Filaris Supravanale.
2. Chonobosis Tuberculosa auriculac corcet et
   tuberculosis milia cor universalis.
3. Cirrhosis et Degeneration amyloidica Hepatis.
4. Hydrops ascites.
5. Degeneration mus alcoholica multiplex
   nervorum.
Remarks on Cases 24 and 55.

The rapidly fatal course and the absence of marked, sensory signs are more particularly noteworthy in Case No. 24, in both cases, the cardiac affection probably due to disease of the Vagi will be noticed and again in both cases, one finds Pulmonary Phthisis. In case No. 55 the change in the Medulla Oblongata were not very pronounced as in the last case possibly to be explained by the relatively short duration of the disease.

In conclusion it will be observed that in all my cases considerable changes have been found in the Spinal Cord, and I may mention that in another case which clinically more resembled to be Dorsal's but of which I have unfortunately not had time to complete. Thus I have found even more marked changes.

In account of this case I hope to publish later, along with the cases which I have in this thesis described.